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Editorial

An Approach to Psychiatric Disorders in Temporal Lobe Epilepsy

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In this commentary we will try to approach psychiatric disorders in patients with temporal lobe epilepsy, which can be underdiagnosed and not always identified, without being able to carry out an adequate therapeutic management, or to improve the quality of life of these patients.

According to the World Health Organization (WHO), epilepsy is one of the most frequent neurological disorders, which is considered a health problem. Recently, it is estimated that more than 70 million people in the world suffer from it, of which about 7 million live in Latin America and the Caribbean [1].

Most epileptic seizures correspond to temporal lobe epilepsy (TLE), which is the most common form in adults, representing between 25% and 35% of general epilepsy cases [3], whose anatomical abnormality is more common is hippocampal sclerosis [2,3].

The definition of temporal lobe epilepsy was established after the lobectomy performed by Penfield in 1954, later it was modified by Walker in 1967 and Falconer in 1979 [4].

According to the International League Against Epilepsy (ILAE) classification [5], TLE can be divided into mesial temporal lobe epilepsy (mTLE) and lateral or neocortical temporal lobe epilepsy (nTLE). mTLE is the most common subtype and seizures originate from the hippocampus, entorhinal cortex, amygdala, and parahippocampal gyrus. The brain structures involved in nTLE are temporal neocortex that includes the superior, medial, and inferior temporal circumvolutions, the temporal-occipital and temporal-parietal junctions and the associative sensorial areas for hearing, visual, and language functions [6].

However, the complexity of the neural networks involved in temporal lobe seizures, more recently allowed the classification into five subtypes according to anatomical location: mesial, temporopolar, mesiolateral, lateral and temporal ¨plus¨. Temporal lobe epilepsy plus is defined as a primary epileptogenic zone of the temporal lobe, extending to neighboring regions such as the insula, suprasylvian opercular cortex, orbitofrontal cortex, and the temporoparietooccipital junction [7].

Clinical manifestations

The age at seizure onset for mTLE is lower than nTLE (10.9 years and 23.2, respectively). A personal history of febrile convulsion is more frequent in mTLE [3].

Temporal lobe epilepsy seizures are characterized mainly by behavior arrest and impaired awareness. Focal aware seizures are frequently reported prior to seizure onset. Autonomic or visceral-sensory seizure, characterized by the abdominal or epigastric rising sensation, is more commonly present in mTLE. Other autonomic manifestations are pallor, flushing, cyanosis, alterations in cardiac frequency and rhythm, vomiting, urinary urgency, piloerection or pupillary alterations [6].

Cognitive and emotional seizures are peculiar of TLE and are characterized by I) dysmnesic features such as déjà vu or jamais vu, II) cognoscitive features with a misperception of internal or external reality, III) emotional features such as panic attacks and behavioral changes, IV) illusions and hallucinations that might set visual, auditive, or olfactory auras [8].

Focal to bilateral tonic-clonic seizures may occur in about 60% of patients with TLE [6].

Oroalimentary automatisms and manual stereotypes movements are described in 40-80% of TLE. Particularly, oral alimentary automatism can be associated with amygdala and anterior temporal región involvement [8].

Ictal speech, characterized by intelligible, well-articulated, linguistically correct language during crisis, is observed especially in non-dominant TLE, whereas in dominant TLE is more frequent to observe ictal aphasia and verbal automatisms [8].

Unilateral dystonic posturing is more common in mTLE [3].

The head deviation is common but not exclusive of TLE with supra-Sylvian structures and frontal lobe involvement [9].

Postictal period is commonly present with a lower frequency in nTLE compared with mTLE (23.5 vs. 85%) and it is characterized by a confusional state sometimes combined with language alterations or psychiatric symptoms [6].

Psychiatric symptomatology

Epilepsy is a chronic neurological condition that is highly predisposed to a variety of mental health problems due to its huge biological, social and psychological burdens [10].

The incidence of neurobehavior disorders is considered to be higher in patients with epilepsy and a relationship can be established between these disorders and focal temporal and frontal lobe epilepsy and in treatment-refractory patients with epilepsy, according to some authors in up to 40% of of the patients, reporting series of patients with epilepsy in which about 70% had a concomitant psychiatric disorder [2].

A study was conducted by Wubie., *et al.* to evaluate the prevalence and associated factors of common mental disorders among people with epilepsy in Ethiopia, 2019, which was high (35.8%), suggesting that it is a problem of public health [10].

Early diagnosis-and consequent management-of psychiatric symptoms in TLE patients is desirable, since it might lead to a better epilepsy outcome, both in terms of quality of life and of healthcare costs [11].

In addition to neurological features, Temporal lobe epilepsy (TLE), is frequently associated with psychiatric disorders [12], which mainly occur in the form of depression and generalized anxiety disorders [13].

Despite being frequent and important, however, mental disorders are underdiagnosed in patients with this disease, the causes of which are usually: a tendency to minimize symptoms; difficulty recognizing unusual, atypical symptoms in the population with epilepsy; tendency on the part of patients to minimize complaints for fear of being discriminated against and fear that psychotropic drugs lower the seizure threshold [14].

These manifestations are often misunderstood with a consequent reduction of quality of life of the patients. While ictal disorders are directly related to seizures, interictal psychiatric disorders may occur independently in the context of epilepsy [15].

Mesial temporal sclerosis (MTS) is correlated to a higher prevalence of psychiatric symptoms up to 70% in pharmacoresistant forms of TLE [6].

Anxiety, depression, and interictal dysphoria often combined with cognitive, learning, and behavioral impairment are recurrent psychiatric disorders in the pediatric patients with TLE [16].

However, acording to most authors, depression associated with epilepsy is the most common psychiatric disorder among people with epilepsy [17].

Andrade-Machado., *et al.* conducted an investigation to study the frequency of suicidal ideation in 82 patients with refractory focal epilepsy and found that 31.7% had severe risk and this type of risk only appeared in patients with temporal lobe epilepsy [18].

Filho., *et al.* [19] in a study of 170 patients with mTLE detected mood disorders in 25.8% of cases followed by the psychotic disorders (15.8%) and anxiety disorders (14.1%). Whereas, according to Ertem., *et al.* [20], anxiety disorders resulted the most common psychiatric comorbidity (23%), followed by mood disorders (17%), psychotic disorders (13%), and somatoform disorders (3%). Seizures worsening and the increasing of seizure frequency are risk factors for psychopathology. Polytherapy with antiseizure medications (ASMs) has been associated with a major risk of behavioral and emotional features. Moreover, familiarity with psychiatric disorders and family disruption are predictors of psychopathology. Hippocampal sclerosis can be associated with an increased risk of frontotemporal network dysfunction resulting in the psychiatric comorbidity. Psychiatric symptoms can occur before and after seizure. Premonitory symptoms, occurring at least 30min before a seizure, are frequently described as irritability, depression, headache, "euphoria," and confusion [21].

About 44% of children with TLE are considered at-risk for depression, while 22% are considered in the clinical significative range [22].

Depression and TLE are supposed to have similar physiopathology with common involvement of hippocampus, amygdala, and long-range frontal lobe projections [22].

The amygdala is determinant in the experience of fear and its autonomic and endocrine responses. Instead, the connection between the amygdala and periaqueductal gray is implicated mainly in avoidance behavior and fear responses. The hippocampus is important in the re-experiencing of fear. Activation of fear circuits is a major hypothesis for explaining symptoms in anxiety disorders [23].

MRI volumetric studies have found decreased volumes of the amygdala and hippocampus in the recurrent and chronic untreated depression [24].

Increasing amygdala volumes, particularly on the left side, are associated with depression severity among patients with TLE [25].

These findings may be due to enhanced regional blood flow and vascular volume as detected by the positron emisión tomography (PET) [26] or secondary to dendritic remodeling with increased branching of amygdaloid neurons [27].

Patients with TLE and aggressive episodes, such as interictal dysphoric depression manifestation, had a decrease of gray matter mainly in the left frontal lobe [28].

The risk of psychosis in patients with epilepsy has been described, which can be 6-12 times more than the general population, with a prevalence of around 7-8%. In patients with treatment-refractory temporal lobe epilepsy, the prevalence has been reported in a range of 0-16% [29].

They can be classified according to their relationship with the occurrence of epileptic seizures in periictal (preictal, ictal or postictal) and interictal [30].

The etiology and pathogenesis of psychosis in epilepsy is still poorly understood, however, neuroanatomical changes have been observed in patients with psychosis [31].

In general, patients with temporal lobe epilepsy and psychosis have significantly smaller brain volume than patients with TLE only.

Attention deficit hyperactivity disorder (ADHD) is a common comorbidity of childhood epilepsy, but the neuroanatomical correlation of ADHD with epilepsy has yet to be comprehensively characterized. High frequency of seizures and nocturnal crisis may alter attention during the day and worsen ADHD symptoms. In patients with TLE, alterations in attentional control may be charged to structural abnormalities outside the temporal lobe involving frontostriatal connections [32].

The greatest deficits appear in divided attention, selective attention, and set shifting that requires a high level of processing resources. In contrast, sustained attention is less compromised and dual-task performance appears to be normal in the patients with TLE [33]. Cognitive and behavioral disorders occur in almost 50% of patients with dysphoric symptoms and usually begin within 24 - 72h postictally [21].

Autism spectrum disorders (ASDs) are frequent comorbidities in childhood and adolescent epilepsies. According to Chez., *et al.* 60.7% of children with ASD present epileptiform activity in sleep frequently localized over the right temporal region [34].

The ILAE estimates an overall prevalence of ASD in the epileptic population of ~20%, whereas the prevalence in the general pediatric population is ~1% [35].

The prevalence of ASD is the highest in cases with epilepsy accompanied by intellectual disability [36].

Children with ASD and epilepsy have greater motor difficulties, developmental delays, and behavioral problems than ASD cases without epilepsy [37].

The causes of ASD are extremely variable and are sometimes common to epilepsy [38].

Increasing evidence suggests that common genetic abnormalities may be associated with both epilepsy and autism [39].

According to Keown., *et al.* [40] the enhanced local visual processing in the autistic patients may be due to the enhanced local connectivity in primary visual and extrastriate cortices, extending into the temporal lobe. Hyperconnectivity of the mesial temporal lobe has as well-been described in TLE [41].

A diagnosis challenge is represented by psychogenic non-epileptic seizures (PNES), which are conversion disorder that is often misdiagnosed in 5-33% of patients considered affected by refractory epilepsy [42].

PNES mainly affect adults but can also occur in children, especially in patients with a history of multiple psychiatric diagnoses [43].

Early recognition of psychiatric features is the first step to guarantee the correct management of this disorder. It is necessary to exclude that symptoms are a consequence of seizures or an adverse effect of antiepileptic medications (AM). For the most AMs, remarkably few studies providing robust data on the psychiatric adverse effects in epileptic patients were identified. Barbiturates, Topiramate, Valproate, and Zonisamide have been reported to cause worsening attention [44].

So too, adverse psychiatric events, including symptoms of depression and anxiety, have also been reported with the use of some antiepileptic drugs (AEDs), particularly barbiturates (Phenobarbital and Primidone), Topiramate, Tiagabine, Zonisamide, Vigabatrin and Leviteracetam [45].

In conclusion, there is evidence of psychiatric disorders in patients with temporal lobe epilepsy, which has a very varied symptomatology. However, neurobehavioral symptoms, which can affect the quality of life of patients and not allow an improvement in seizure semiology, should not be ignored or minimized, nor can the patient be fully managed.

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